

1

FILE COPY

AD-A212 699

SOURCES OF ASTHENOPIA IN NAVY FLIGHT SIMULATORS

by

Sheldon M. Ebenholtz

Institute for Vision Research  
SUNY/College of Optometry  
100 E. 24th Street  
New York, N.Y. 10010

DTIC  
ELECTE  
SEP 11 1989  
S D  
CB

DISTRIBUTION STATEMENT A

Approved for public release;  
Distribution Unlimited

for

Naval Training Systems Center

April 25, 1988

Contract No. DAAL03-86-D-0001  
Delivery Order 0679  
Scientific Services Program

The views, opinions, and/or findings contained in this report are those of the author and should not be construed as an official Department of the Army position, policy, or decision, unless so designated by other documentation.

89 9 8 006

## REPORT DOCUMENTATION PAGE

Form Approved  
OMB No 0704-0188  
Exp Date Jun 30 1986

1a. REPORT SECURITY CLASSIFICATION			1b. RESTRICTIVE MARKINGS		
2a. SECURITY CLASSIFICATION AUTHORITY			3. DISTRIBUTION/AVAILABILITY OF REPORT <del>May not be released by other than sponsoring organization without approval of US Army Research Office.</del>		
2b. DECLASSIFICATION/DOWNGRADING SCHEDULE			5. MONITORING ORGANIZATION REPORT NUMBER(S) TCN 87-635		
4. PERFORMING ORGANIZATION REPORT NUMBER(S) Delivery Order 0679			7a. NAME OF MONITORING ORGANIZATION U.S. Army Research Office		
6a. NAME OF PERFORMING ORGANIZATION Dr. Sheldon M. Ebenholtz		6b. OFFICE SYMBOL (If applicable)		7b. ADDRESS (City, State, and ZIP Code) P.O. Box 12211 Research Triangle Park, NC 27709-2211	
6c. ADDRESS (City, State, and ZIP Code) 29 Heritage Drive Edison, NJ 08820		8a. NAME OF FUNDING/SPONSORING ORGANIZATION Naval Training Systems Center		8b. OFFICE SYMBOL (If applicable) Code 711	
8c. ADDRESS (City, State, and ZIP Code) LCDR Michael G. Lilienthal Human Factors Division Orlando, FL 32813-7100		9. PROCUREMENT INSTRUMENT IDENTIFICATION NUMBER			
11. TITLE (Include Security Classification) Sources of asthenopia in Navy flight simulators.		10. SOURCE OF FUNDING NUMBERS			
		PROGRAM ELEMENT NO.		PROJECT NO.	TASK NO.
					WORK UNIT ACCESSION NO.
12. PERSONAL AUTHOR(S) Sheldon M. Ebenholtz					
13a. TYPE OF REPORT FINAL REPORT		13b. TIME COVERED FROM 22Sep'87 to 25Apr'88		14. DATE OF REPORT (Year, Month, Day) 1988, April, 25	
15. PAGE COUNT 11					
16. SUPPLEMENTARY NOTATION Task was performed under a Scientific Services Agreement issued by Battelle, Research Triangle Park Office, 200 Park Drive, P.O. Box 12297, Research Triangle Park, NC 27709					
17. COSATI CODES			18. SUBJECT TERMS (Continue on reverse if necessary and identify by block number)		
FIELD	GROUP	SUB-GROUP	Asthenopia, eye strain, headache, nausea, oculomotor systems, flight simulators, retinal slip signal, optokinetic nystagmus (OKN), vestibulo-ocular reflex (VOR), pursuit suppression of (over)		
19. ABSTRACT (Continue on reverse if necessary and identify by block number) Asthenopia was defined and the oculomotor systems responsible for eye strain were identified. The oculomotor systems functional during flight simulator training also were described and the conditions under which Navy flight simulators could produce asthenopia were pointed out. Asthenopic symptoms are closely related to those of simulator sickness. In order to lower the incidence of asthenopic symptoms the verification of the proper calibration of virtual-image optics by application of computerized ray-trace analysis was recommended along with additional research efforts.					
20. DISTRIBUTION/AVAILABILITY OF ABSTRACT <input checked="" type="checkbox"/> UNCLASSIFIED/UNLIMITED <input type="checkbox"/> SAME AS RPT <input type="checkbox"/> DTIC USERS			21. ABSTRACT SECURITY CLASSIFICATION		
22a. NAME OF RESPONSIBLE INDIVIDUAL			22b. TELEPHONE (Include Area Code)		22c. OFFICE SYMBOL

18 (continued)

VOR and OKN, accommodation, vergence, dark focus, dark vergence, heterophoria,  
individual differences:

## Introduction

### Oculomotor systems functional in simulator viewing.

All oculomotor systems are active in simulator viewing. Table 1, below, represents a listing of these systems along with their major function and stimulating conditions. Most are well known but evidence that expanding and shrinking patterns may actually modulate accommodation (Kruger and Pola, 1985) is relatively new. Such patterns are capable of mediating depth perception (Beverley and Regan, 1983) and hence the possibility of a pattern-driven vergence response also is indicated.

With the exception of accommodation all of the remaining systems utilize the same two sets of six extra ocular muscles. Hence when more than one demand for eye movement is present, systems may compete for eye movement control resulting in conflict among oculomotor systems. This in turn will produce retinal error or slip signals, which because of the prevalence of negative feedback control will result in compensatory error-correcting eye movements. According to available clinical evidence and theoretical developments the presence of slip signals sustained over moderate time periods is likely to produce asthenopia. The potential sources of slip signals in flight simulators and their relation to asthenopia will be more fully developed below.

### Asthenopia.

#### a) Definitions and types.

The term was first used by MacKenzie (1843) to mean, literally, "eye without strength" and therefore an eye that was strained even in normal use. Early visual scientists and physicians initially identified eye strain with problems of focusing (e.g., Donders, 1864), and hence with ametropia, e.g., myopia (near sightedness), hyperopia (far sightedness) and presbyopia, a condition of aging in which the lens lessens its normal contractility. At about the same time muscular imbalance and heterophoria (Stevens, 1887; Plooge and Howell, 1965) also were recognized as potential sources of asthenopia. It is important to note that the term "heterophoria" refers to the fundamental condition wherein the two eyes fail to point in the same direction in space, except for the operation of a special error correcting loop, the fusion reflex. Thus in heterophoria single vision is maintained under stress. Figure 1 below illustrates a method of measurement of a lateral heterophoria by the use of dissimilar targets that fail to trigger the fusion reflex. Slight reflection on the fact that adequate binocular vision requires the coordination of two sets of six extraocular muscles, plus the pair of intraocular ciliary muscles, by three separate cranial nerves, leads readily to the possibility of oculomotor malfunction and stress.



Dist		Av. and/or Special	
A-1			

50  
Codes

Table 1

Oculomotor control systems operational while viewing kinetic virtual image displays

<u>Type of stimulation</u>	<u>Primary function</u>
optical	single vision
	clear vision
	gaze stability
inertial	single vision
	clear vision
	gaze stability

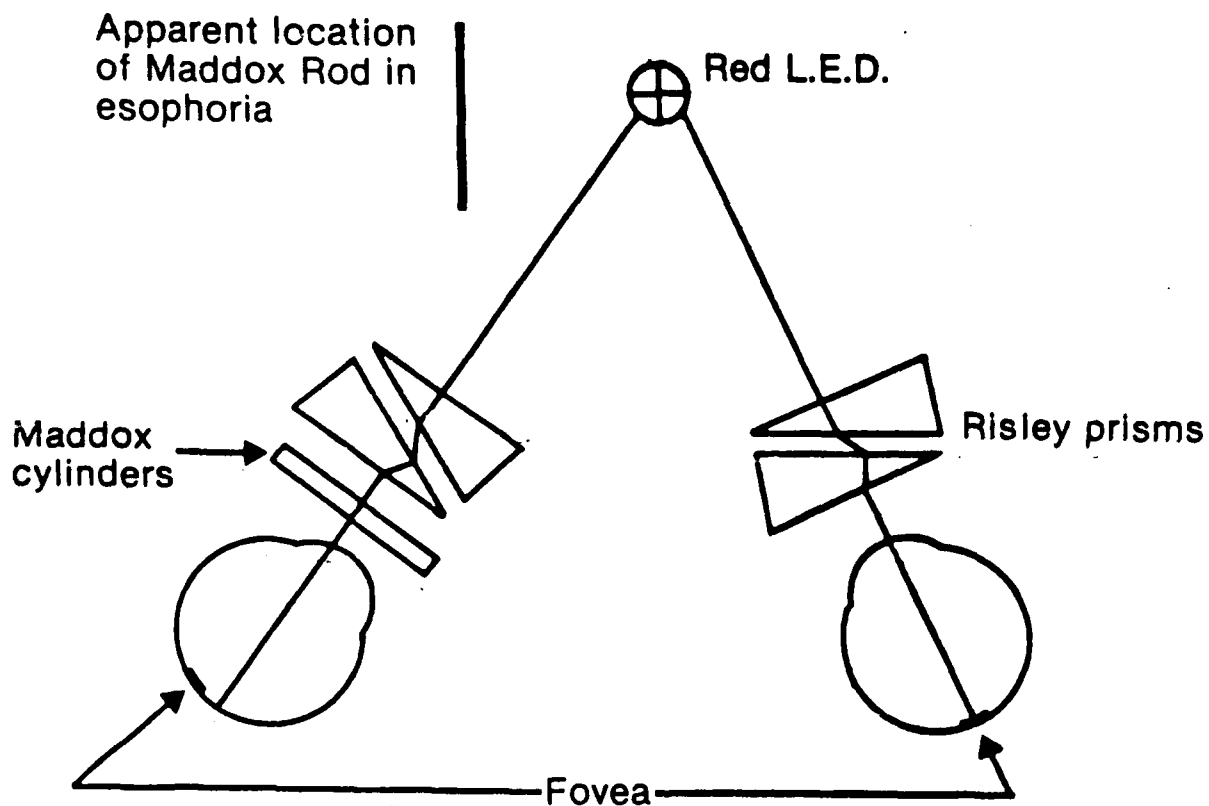


Figure 1. A method of measurement of lateral heterophoria by disassociation of the two eyes. The Maddox cylinders focus the small light-emitting diode (L.E.D.) as a line on the retina while the Risley prisms over the left eye permit the line-image to be shifted to the fovea so as to appear coincident with the image of the L.E.D. The amount of prism shift is equivalent to the heterophoria magnitude.

In more recent times, (Ames, 1935; McKee and Provines, 1987) aniseikonia, a condition of unequal image sizes in the two eyes, has been recognized as a source of asthenopia, and a pupillary origin also has been proposed (Cowan, 1955).

#### b) Symptoms.

Purely visual symptoms such as diplopia (double vision) or patent blur are not reliable indicators of the onset of eye strain because "...the condition is caused essentially by the effort to compensate for optical and muscular imperfections and if such compensation is impossible, no sustained effort is attempted." (Duke-Elder, 1970, P. 566). It is thus the small errors which the oculomotor systems are capable of correcting that lie at the source of asthenopia. It is only when these systems fail that large scale errors in control will be manifest in consciousness in the form of blur, double images and illusions of movement but, paradoxically, in the absence of compensatory processes, the latter events may not produce the pain of asthenopia. Furthermore, although ocular pain associated with extraocular and intraocular muscle is a significant characteristic of asthenopia, it would be wrong to assume the cause to be similar to that of skeletal muscle fatigue and the muscle cramp that ensues with further exercise: physiological fatigue of the extraocular muscles is rare and even spasm of the ciliary muscle usually is painless. There is thus, a clear gap in our understanding of the relation between ocular pain and ocular muscle control.

Asthenopic pain may be located in the orbits or more widespread as a general headache, and may implicate the neck and the eye brows as well. Further symptoms include the sensation of heavy eyelids and drowsiness, hyperaesthesia of the scalp (Duke-Elder, 1970, P. 570), vertigo, and gastric disturbances including indigestion, dyspepsia, nausea, and vomiting.. It is important to note that asthenopic symptoms form a significant subset of symptoms characteristic of the simulator-sickness complex (Kennedy et al., 1987, P. 9).

### Discussion

#### Theories and explanations of asthenopia.

There are no theories of asthenopia in the sense of a body of propositions from which, together with a set of initial conditions, one may logically deduce the asthenopic symptoms. A list of aetiological factors, however, such as those implicated in the various types of asthenopia along with environmental and individual predisposing conditions may serve to point the way to a statement of principles.

Table 2, below, based in large part on the analysis of Duke-Elder and Abrahms (1977) represents a set of conditions known to produce Asthenopia.

Table 2

Conditions Productive of Asthenopia

1. Environmental factors.

- a) level of target luminance and contrast between target contour and surround
- b) size of object detail or its spatial frequency
- c) velocity characteristics of the target and its background

2. Ocular and oculomotor factors.

- a) uncorrected ametropia: hyperopia, myopia, astigmatism, anisometropia
- b) accommodative dysfunction including infacility and excessive accommodation
- c) heterophoria beyond normative levels
- d) convergence excess and infacility
- e) excesses or inadequacies in the ratio of accommodative convergence to the unit change in accommodation (AC/A), and in ratio of convergent accommodation to the unit change in convergence (CA/C)
- f) aniseikonia

3. Individual predisposing factors.

- a) physical fatigue and exhaustion
- b) insufficient sleep
- c) emotional strain

4. Other potential factors.

- a) level of accommodative demand relative to the resting level of accommodation or dark focus (DF)
- b) level of vergence demand relative to the resting level of vergence or dark vergence (DV)
- c) degree of adaptability or plasticity of accommodation and vergence systems
- d) the degree to which the oculomotor systems supporting fixation and pursuit are capable of suppressing vestibular and optokinetic nystagmus respectively.



Table 2, above, is of course not all inclusive but it does cover the major presently known asthenopic conditions. Analysis of these diverse conditions suggests that it is a form of oculomotor instability, in the sense of a control system that is constantly being perturbed, that is a major underlying cause. The view that conflict and instability underlie asthenopia has been promulgated since Lancaster (1932) "...suggested that the strain lay, not in the total expenditure of muscular effort, but in constantly shifting and changing adjustments of the intra- and extra-ocular musculature in futile groping after a more satisfactory but unattainable ideal" (Duke-Elder and Abrahms, 1970, p. 561). Duke-Elder and Abrahms (1970) also expressed their own similar view noting that when the visual "...error is small the patient is able to rectify it to a greater or less extent by muscular effort; this he continually attempts to do to the best of his ability, and the constant strain thus unconsciously imposed upon him brings on muscular and nervous fatigue with its attendant train of reflex symptoms. It is not the error itself which causes the trouble so much as the continuous effort called forth automatically in the attempt to correct it." (p. 564). An essentially isomorphic view was developed independently by Ebenholtz (1986) in an account of adaptive oculomotor control systems. The initial condition both for adaptation and dysfunction, including asthenopia, was the continual triggering of negative feedback loops in an effort to overcome visual and oculomotor error, while the conflict among oculomotor systems was shown to be the source of this error.

#### Application of conditions and theories of asthenopia to Navy flight simulators.

The type of simulator to which the following observations most directly apply is the concave mirror-CRT virtual-image device or any other device with equivalent optics.

a) Of the three environmental factors, all of which apply, item 1c has the greatest potential for asthenopia since it is not covered by explicit standards. Background velocity represents a parafoveal stimulus to optokinetic nystagmus (OKN) in which the oculomotor system attempts to match eye velocity with retinal image velocity. Likewise, the foveal target also triggers the foveal pursuit system with the same goal. If velocities of either the background or the target exceed system capabilities a velocity error signal will be generated which could, if sustained lead to strain. Furthermore if background and target velocities are in different directions, the suppression of OKN must be adequate. If otherwise, velocity error signals again will be generated (Yee et al. 1983) along with the potential for eye strain symptoms.

b) The ocular and oculomotor factors of Table 1 refer essentially to parameters of individual oculomotor control systems. In virtual image displays, however, it is possible for improperly calibrated systems to make stressful demands on the vergence and accommodative systems of otherwise visually normal observers. Thus, e.g., displays producing converging beams that form real images at

or behind the design eye are likely to yield visual stress similar to convergence excess. Perhaps less widely known, however, is the fact that lateral displacements even as small as 9 inches, from the design eye are capable of producing both unequal accommodative demand (i.e., induced anisometropia) and unequal retinal image sizes, i.e., induced aniseikonia (see interim report, this delivery order). Both of these conditions are known to produce eye strain symptoms.

c) The individual predisposing factors of Table 1 are generally known and will not be amplified here.

d) Other potential factors listed in Table 1 derive from the relatively recent knowledge of the resting levels of accommodation (Leibowitz and Owens, 1978) and of vergence respectively (Owens and Leibowitz, 1980). These have been shown to be at intermediate distances, frequently within a few feet of the observer, and not at optical infinity as previously thought, except for specific individual differences. Since the degree of work or effort required for focusing or for verging will vary directly with the distance of the target from the resting level, observers having distant resting levels are required to do less work for stimuli at or near optical infinity, than observers with closer resting levels. Since the purpose of virtual image devices is to produce images at optical infinity, observers with distant resting levels are less likely than others to be candidates for asthenopic symptoms under conditions requiring sustained distant viewing.

A second issue also based on recently acquired knowledge derives from the fact that the resting levels of accommodation and vergence are capable of adaptation in the sense of a shift in the direction of the sustained accommodative or vergence demand (Ebenholtz, 1981; 1983). There is, however, great variability among individuals in the magnitude and direction (e.g., near or far of such adaptive capability (e.g., Ebenholtz, 1985; Schor, 1979). The significance of this capability lies in the accumulating evidence that individuals who exhibit asthenopic symptoms are more likely to have non-adaptive or slowly adapting vergence and accommodation systems than are asymptomatic observers (Fisher et al., 1987; North and Henson, 1981).

Finally, little empirical data exist concerning the relation between asthenopia and the ability to suppress vestibular and optokinetic nystagmus in order to prevent retinal slip during pursuit. It remains therefore, as a logical possibility that individuals with poor suppression capability will be more susceptible to asthenopia than those with more capable systems. Furthermore, it is important to add that in simulators used with a motion base there is the likelihood that the vestibular-ocular-response (VOR) will be triggered along with the optokinetic response, though not necessarily simultaneously. In these simulators there exists therefore, an increased probability of retinal slip since two potential sources are available to trigger retinal error. One may expect therefore, that in simulators with a motion base, an enhanced rate of simulator sickness is to be expected, relative to

simulators used without the motion base, all else held constant.

## Conclusions

### Recommendations for future research and development with application to flight simulator design specification.

#### a) Information required for ray-tracing analysis of convergence and accommodation.

The need to have accurate scaled drawings of the layout of all the optical components of the virtual image display system has been noted previously in an interim report dated March 11, 1988. In addition to a computerized ray-trace analysis of the light vergence from each window (i.e., each concave-mirror-CRT system), a field measurement of light vergence should be made from the area of the design eye using an instrument such as a precision dioptometer. In general, converging light bundles are not acceptable and differences in light vergence between windows should be minimal, less than .12 diopters. Likewise differences in light vergence between each eye should not exceed .06 diopters.

It may be noted here that as the number of "windows" i.e., concave mirror-CRT systems, increases in any given simulator, there is an increased risk of miscalibration. This is specially true of the placement of the CRT at the appropriate distance from the mirror, since converging light beams result from placing the CRT beyond one focal length of the system. The need to maintain scene continuity without gaps, and horizontal patterns without spatial discontinuity also increases the risk of calibration error as the number of windows increases. Accordingly, future simulator designs should move toward lowering the risk of calibration error by minimizing the number of required CRT-mirror systems in a given simulator.

#### b) Standards for contrast and luminance levels of moving scene images.

Poor contrast between edges and backgrounds is a source of stress for the accommodative system, since the focusing system normally works to increase contrast by reducing blur. Research is needed to determine dynamic contrast sensitivity functions for moving targets, as a function of spatial frequency and target velocity. Such standards would enable evaluation of various computer generated graphics displays with respect to adequacy of dynamic contrast.

#### c) Standards for optokinetic velocities.

The simulated displays in any given scenario vary in the extent of visual stress. Research is needed to verify those aspects of scene content that are most conducive to eye strain. For example, velocity of the images measured at the observer's eye

is a likely factor since the optokinetic response is velocity sensitive. If the strength of the OKN is too great to be fully suppressed, then retinal slip signals will occur and asthenopic symptoms will follow. Research should be aimed at determining those characteristics of the scene, such as texture density and image velocity that are likely to modulate the strength of the OKN. This information may be particularly useful in the creation of guidelines for the development of low altitude flight scenarios where display velocity and texture density are likely to be high enough to create eye strain. For the same reasons, helicopter flight trainers also are likely to be especially susceptible to optokinetic eye strain and asthenopia.

d) Field studies during flight simulator training.

There is a significant gap in knowledge of how eye movement systems actually perform during flight simulator training. This gap should be remedied by a research program with the goal of measuring the acceleration and velocity profiles of head movements and concurrent eye movements while simultaneously recording the velocity profile of the visual images of the scene to which the pilot is responding. Additional research is needed to describe the actual vergence and accommodation responses to the known light vergence from the scene. Furthermore the understanding of individual differences in response to potentially asthenopic stimuli would be aided by measurement of a) individual capability to suppress nystagmus (both OKN and VOR), b) the resting level of accommodation and of vergence, and c) the lateral heterophoria at near and far distances.

## REFERENCES

- Ames, A. Jr. (1935). Aniseikonia - A factor in the functioning of vision. Amer. J. Ophthal., 18, 1014-1020.
- Beverley, K.I., and Regan, D. (1983). Texture changes versus size changes as stimuli for motion in depth. Vision Research, 23, 1387-1400.
- Cowan, A. (1955). The role of the pupil in ametropia. American Journal of Ophthalmology, 40, 481-485.
- Donders, F.C. (1864). On the anomalies of accommodation and refraction of the eye. Translated by W. D. Moore, London, New Sydenham Society.
- Duke-Elder, S. and Abrams, D. (1970). Eye-strain and visual hygiene, Ch. 15 in Duke-Elder, S. (Ed.) System of Ophthalmology, Vol. 5. C.V. Mosby, St. Louis.
- Ebenholtz, S.M. (1986). Properties of adaptive oculomotor control systems and perception. Acta Psychologica, 63, 233-246.
- Ebenholtz, S.M. (1985). Accommodative hysteresis: relation to resting focus. Amer. J. Optom. Physiol. Opt., 62, 755-762.
- Ebenholtz, S.M. (1983). Accommodative hysteresis: a precursor for induced myopia? Invest. Ophthalmol. Vis. Sci., 24, 513-515.
- Ebenholtz, S.M. (1981). Hysteresis effects in the vergence control system: Perceptual implications. In Eye Movements: Visual perception and cognition. D.F. Fisher, R.A. Mority, and J.W. Senders (Eds.), Lawrence Erlbaum and Associates, Hillsdale, New Jersey.
- Fisher, J.K., Ciuffreda, K.J., Levine, S. and Wolf-Kelly, K.S. (1987). Tonic adaptation in symptomatic and asymptomatic subjects. Amer. J. Optom. Physiol. Opt., 64, 333-343.
- Kennedy, R.S., Berbaum, K.S., Lilienthal, M.G., Dunlap, W.P., Mulligan, B.E., and Funaro, J.F. (1987). Guidelines for alleviation of simulator sickness symptomatology. Orlando, Fl.: Naval Training Systems Center, NAVTRASYSCEN TR-87-007.
- Kruger, P.B. and Pola, J. (1985). Changing target size is a stimulus for accommodation. Journal of the Optical Society of America A, 2, 1832-1835.
- Lancaster, W.B. Ocular symptoms of faulty illumination. American Journal of Ophthalmology, (1932). 75, 783-788.

Leibowitz, H.W. and Owens, D.A. (1978). New evidence for the intermediate position of relaxed accommodation. Documenta Ophthalmologica, 46, 133-147.

MacKenzie, W. (1843). On asthenopia or weak-sightedness. Edinburgh Medical and Surgical Journal, 60, 73-103.

McKee, M.C. and Provines (1987). Nausea caused by Aniseikonia. Amer. J. Optom. Physiol. Opt., 64, 221-223.

North, R. and Henson, D.B. (1981). Adaptation to prism-induced heterophoria in subjects with abnormal binocular vision or asthenopia. Amer. J. Optom. Physiol. Opt., 58, 746-752.

Owens, D.A. and Leibowitz, H.W. (1980). Accommodation, convergence, and distance perception in low illumination. Amer. J. Optom. Physiol. Opt., 57, 540-550.

Ploudre, A.R. and Howell, W.H. (1965). Lateral heterophoria: symptoms, measurement and treatment. Journal of the American Optometric Association, 36, 811-817.

Schor, C.M. (1979). The influence of rapid prism adaptation upon fixation disparity. Vis. Res., 19, 757-765.

Stevens, G.T. (1887). The anomalies of the ocular muscles. Archives of Ophthalmology, 16, 149-176.

Yee, R.D., Daniels, S.A., Joles, D.W., Baloh, R.W., and Honrubia, V. (1983). Effects of an optokinetic background on pursuit eye movements. Invest. Ophthalmol. Vis Sci., 24, 1115-1122.